PHYSIOLOGICAL EFFECTS OF AMMONIA.—O. Funke and A. Deahna, *Pflueger's Arch. f. d. ges. Phys.*, IX., s. 416. In frogs the authors noticed, on administration of ammonia in any way whatsoever, the occurrence of tetanic convulsions, following the primary manifestations of pain, after a time proportionate to the concentration of the article. This spasm, which the authors consider reflex in its nature, is soon followed by utter prostration, accompanied, however, by a state of *exalted* reflex irritability, so that the slightest sensory impression, even loud shouting, is responded to, though feebly, by reflex movements. Still a repetition of the tetanus was never observed, probably from the great exhaustion. In other more severe cases, which pursue a fatal course, reflex irritability is entirely destroyed by the primary tetanus, while motor nerves and muscles still respond to a direct stimulus. Rabbits react in a similar manner on injection of the agent into the blood; the hypodermic method was found of much less effect in them.

The tetanus is of centric origin, as it implicates the posterior extremities even after their blood supply is cut off, while they escape the convulsions on section of the sciatic nerves. Division of the cord below the medulla does not affect the result. The reflex impulses are so powerful as to overcome even the paralysis induced by moderate doses of curare. In short, the agent appears to be identical in its action on the cord with strychnia.

The effects of ammonia on the circulation are a feeble reduction of the blood-pressure, followed in a short time by an enormous increase of the same, while from the beginning the frequency of the pulse is diminished. Both the latter and the primary diminution of pressure were found to be the result of a strong excitation of the vagus of centric origin, though it could not be determined whether this was due to stimulation of that centre, or merely increase of its reflex irritability. The secondary augmentation of tension is caused by powerful contraction of the systemic arterioles, from excitation mainly of the vaso-motor centre in the medulla, though it was not proven conclusively that the stimulation was limited to this centre alone. The dyspnœa produced by the agent is indicated by a primary acceleration of respiration, which, at first shallow, hereupon becomes deeper. Larger doses produce a subsequent *arrest* for two to three seconds, which is sometimes the only respiratory alteration. If now tetanus occurs, respiration is more or less arrested during the entire period. Hereupon an augmentation, both in frequency and depth of respiration, follows, all of which phenomena are rendered more prominent by section of the vagi. This latter fact would therefore prove that the arrest of respiration is not due merely to an irritation of the peripheral end of the vagus by the agent, which Knoll had previously shown to occur. H. G.

NITRITE OF AMYL.—W. Filehne, *Pflueger's Arch. f. d. ges. Phys.*, IX., s. 470. In order to avoid the fright of the animals (rabbits), as well as any impression of the vapor on the trigeminus-terminations in the nasal mucous membrane, inducing reflex phenomena, the author caused the animals to inhale through a canula in the divided trachea. The question whether the dilatation of vessels caused by nitrite of amyl is due to an influence of the

agent on the musculature of the vessels, Filehne tried to solve by observing the pulmonary vessels, after laying bare the transparent pleura, which he found were not altered in calibre. On severing one sympathetic in the neck, and including the same in an induced current of such an intensity that the vascularity in the corresponding ear did not perceptibly differ from that of the other side, inhalation of the vapor caused hyperæmia only on the normal side. The legitimate deductions from this experiment are that nitrite of amyl paralyzes neither the vessels themselves, nor their nerves, but causes hyperæmia by destroying the tonus of the vaso-motor nerves. This need not be referred necessarily to paralysis of the vaso-motor centres, but may be explained on the theory of reflex inhibition. Section, however, of the *depressor* nerves (Ludwig) does not alter the result.

The heart of the frog was found to become paralyzed by the drug, while, on the contrary, the rabbit's pulse was much accelerated. The latter was counted by introducing into the heart an acupuncture-needle, armed at its free end with a pellet of sealing wax, which, at every systole, struck against a glass tumbler. After every tenth stroke, as counted by the ear, a chalk-mark was made, thus enabling the observer to record even a very frequent beat. The acceleration of the pulse is due to destruction of the tonus of the pneumogastric nerves (which, being not maintained in the frog, accounts for the want of acceleration in that animal). In conclusion, the author refers to the analogy, first pointed out by Darwin, between the effects of nitrite of amyl on animals, and the result of some psychical processes in man, manifested by blushing of certain parts of the body, and an increase in the frequency of the pulse, maintaining that blushing, etc., is not a privilege of man alone, though in animals, on account of inferior mental development, a psychical impulse is wanting, while its place can be supplied by the agent in question. H. G.

THE reaction of nerves to the galvanic current, according to whether the circuit is closed by the cathode or anode, has been studied by H. Engesser (*Pflueger's Arch.*, X., p. 147), since Hitzig asserted he had found a greater sensitiveness to the anode in his experiments on the excitability of the hemispheres. Engesser, however, could not detect any difference in effect in the peripheral nerves, either exposed in the frog or undisturbed in man. H. G.

NITRITE OF AMYL IN TETANUS.—Dr. Wm. S. Forbes read before the College of Physicians of Philadelphia, April 7th (reported in *Med. Times*), the history of a case of acute traumatic tetanus, beginning the fourth day after an extensive burn, which was treated and completely cured in forty-six days by inhalations of nitrite of amyl. Forty hours after the onset of the disease, the temperature was 102, the pulse 133, and the respiration 32, with marked opisthotonus and trismus, considerable involvement of the muscles of nutrition, and painful spasmodic paroxysms. The administration of the nitrite was commenced on the evening of the sixth day after the accident, and when the patient was in the condition above described. The sole treat-